Indian Journal Of Otology
Vol. 16, March 2010, P. 3-7

EDITORIAL

SUDDEN SENSORINEURAL DEAFNESS

It may be defined as an abrupt or rapidly progressing hearing loss greater than 30 dB in three contiguous pure tone frequencies occurring in less than three days duration. Majority of cases are unilateral and all the more idiopathic are almost always unilateral , developing in few hours or suddenly on waking up, or after attending a phone call. Bilateral involvement is rare.

In majority cases or studies no sexual, racial or geographical prediliction could be found, though most cases are observed above the age of forty years but sudden sensory neural deafness has been reported not infrequently in children also. As per literature available both the ears are equally affected.

Sudden sensorineural deafness is a presenting symptom but in about 50% cases it is associated with either tinnitus, and/or vertigo.

Associated Symptoms

In almost all the cases event is memorable and incidence may be reproduced verbally.

Tinnitus is the most common associated symptom which precedes in about 75% cases from minutes to hours before the development of deafness. It may be roaring to steam engine type common cricket type of sound is usually not reported.

The presence of tinnitus is supposed to be a favorable prognostic sign. Pain or feeling of pressure may be present in the affected ear.

It has many possible etiologies:

PATHOPHYSIOLOGY

Four theories are proposed:

Labyrinthine Viral Infection

Seroconversion rates are statistically significantly higher for mumps, rubella, varicella zoster, cytomegalo virus and influenza.[1] There is usually history of recent viral type of illness and evidence of inner ear histopathology is consistent of viral infection present. Out of the above viruses incidence of herpes virus is significantly higher. Sub clinical mumps infection was documented in 9 of 130 patients by positive Immunoglobulin (IgM) mumps antibodies[2]. In known cases of viral labyrinthitis on histopathological examination Schuknecht and Donovan have reported, degenerative changes of tectorial membrane and of stria vascularis and atrophy of organ of corti. Schuknecht et al concluded viral cochleitis was the most possible cause of sudden hearing loss[3]

Vascular Etiology

Logically it is more appealing since cochlea is an end organ in terms of its blood supply having no collateral circulation hence obliteration or reduced blood supply either due to embolus, thrombosis or vasospasm may result in necrosis of membranous labyrinth followed by ossification and fibrosis.

Auto-immune Diseases

The incidence of progressive hearing loss due to syndrome, systemic lupus erythematosus and in auto-immune diseases is documented but sudden deafness is less likely and reversal is more likely. A study by Toubie Et al has well documented the association of multiple immune mediated disorders with idiopathic sudden sensory neural hearing loss.[4]

Membrane Rupture

Rupture of round or oval window has been reported as a cause of sudden hearing loss though most of the patients do not give a positive history of implosive, explosive or blast forces to affect the membranes or membranous labyrinth but somehow Simmons in 1968 has observed association of a popping, clicking sound preceding the hearing loss or a sudden development of a marked tinnitus.[5, 6]

Other causes trauma, toxins (snake bite) tumor specifically acoustic neuroma.

MATERIAL & METHODS

This progressive study was conducted at Indian institute of ear diseases, Delhi & Muzaffarnagar from Jan 2005 to Dec 2009, a total of 244 patients were registered 'exclusion criteria was associated ear diseases viz otitis media or prior tinnitus, vertigo and deafness or any other central nervous system involvement. The pure tone audiometry air conduction was performed at 250 Hz,500 Hz, 1KHz, 2KHz, 4KHZ and 8kHZ and bone conduction at 500 Hz,1KHz,2KHz,4KHZ.

Out of 244 patients 131 male and 113 were females ranging from 10yrs to 71yrs, mean age was 43yrs, all patients were examined for complete E.N.T examination apart from routine general examination and central nervous system examination. Blood examinations were performed including lipid profile. History of drug intake, alcohol consumption, smoking or tobacco chewing was always asked , job profile was also assessed for ototoxicity and sound pollution (acoustic trauma)

Level of Hearing Loss

We used the 1970 classification proposed by Davis and Silverman[7] to define the degree of hearing loss (HL; average of 500, 1,000, and 2,000 Hz): normal, 0-25 dB HL; mild, 26-40 dB HL; moderate, 41-70 dB HL; severe, 71-90 dB HL; and profound, 91 dB HL or higher.

The patients were divided into three groups after explaining the treatment protocol and investigation profile. Since institute is a paid set up all patients were offered a lucrative package of six months in which last four months were free including audiogram inspite of that 27 patient lost to follow up hence not included in this study.

Table-I

Therapy

a) Oral steroids + methyl cobalamine 1500 microgram doses total 89

b) Oral steroids + intra tympanic injection methyl prednisone + methyl cobalamine total 59

c) Oral corticosteroids +intra tympanic methyl prednisone + methyl cobalamine + acyclovir total 96

Inclusion Criteria[8]

- Sudden deafness for up to 2 months.
- Absence of previous treatment
- Absence of alteration of external or middle ear.
- Normal tympanometry curve
- Audiometry with sensorineural hearing loss.
- Normal magnetic resonance image of temporal bone, eliminationg the possibility of tumors or demyelinationg deseases

The evaluation of patients was done as follows:-[9] hearing improvement up to the level of healthy side or less than 20dB HL of the mean level at 6 frequencies, considered as full recovery or cure.

Improvement of more than 10dB in all frequencies leveled as grade II A recovery and less than 10 dB at all frequencies as grade II B was treated as unsatisfactory recovery.

RESULTS

In this study association of smoking & tobacco chewing, diet could not be confirmed. The male-female ratio was not significant, no. of male 131 and female were 113. Out of 244 patients 16 patients were having mild, 69 moderate, 113 severe and 46 had profound deafness.

In associated symptoms, ear blockage which was observed in 164 (67.21%) ears, was associated with tinnitus in 59 (24.18%), vertigo in 12 (4.92%) and tinnitus and vertigo in 28 (11.48%) cases. Vertigo was associated in 84 (34.43%) cases while tinnitus was observed in 163 (66.80%) cases which was solitory in 36, but with vertigo in 40 (16.39%), tinnitus and ear blockage in 59 (24.18%) and tinnitus ear blockage+vertigo in 28 (11.48%) cases. Out of 244 patients 113 patients had severe deafness, 46 (18.85%) had profund hearing loss, 69 had moderate degree of hearing loss and 16 had mild degree of hearing impairment. In severe group 29 patients had cured 33 had good improvement, 29 mild improvement and no improvement was observed in 22 (9.02%) cases. There was over all 57% (23.3%) had cured and 70 (28.69%) had good improvement, 67 slight (mild) improvement and 50 (20.49%) had no improvement, but practically only 127 (52.05%) were satisfied and 117 (47.95%) were not satisfied with treatment.

Age & Sex Wise Distribution of Patients					
Age	Male No.	%	Female No.	%	Total No. %
10-20	18	47.37	20	52.63	38 100
20-45	51	53.68	44	46.32	95 100
45-71	62	55.86	49	44.14	111 100
Total	**131**	**53.69**	**113**	**46.31**	**244**

Table-II

The patient treated with oral corticosteroid with methyl cobolamine 89. 21+24=45 had satisfactory improvement 19+25=44 had no improvement while after adding intratympanic methyl prednisolone 28 (47.46%) had satisfactory improvement and 31 (52.54%) had poor result or no satisfaction. In group C in which acyclorir was also added apart from oral corticosteroids+methyl cobalamine 1500 mcg in 96 patients 54 (56.25%) had satisfactory improvement and 42 (43.75%) had poor response.

Complication of Using IT steroid	
• TM Perforation	2
• Trasient Vertigo	23
• Infection	1
• Acne	1
Total	**27**

DISCUSSION

In our study we could not find out any correlation with smoking, drinking habits, diet, sleeping pattern & time, appetite, environmental sounds and environmental factors including exposure to cold, previous diseases, tiredness, incidence of common cold, hypertension and thyroid disorder etc. While nakashima; et al[10] reported a role of diet and environmental factors.

Level of Hearing Improvement		
Grade	**No.**	**Percentage**
Gr.I	57	23.36%
Gr. II A	70	28.69%
Gr II B	67	27.46%
No Cure	50	20.49%
Total	**244**	**100.00%**

Table -III

In this study we carried out a randomized study no influence of sex could be predicted but there was definite poor prognostic value in older age group but Kronberg; et al[11] & Molini; et al[12] have observed no prognostic significance of age.

In this work it was observed that association of tinnitus, younger age group and low frequency hearing loss were better prognostic sign. High frequency severe hearing loss specifically at 8000 Hz was a bad prognostic sign similar observation has been

observed by De Barros et al[8] and in 1999. Parnes has performed comparative study and observed that methyl prednisolone had the highest levels in the inner ear and sustained higher levels for a longer period of time. And there was a 50% recovery rate.[13]

Sudden Deafness Associated with Other Symptoms		
	No	%
1. Tinnitus	36	14.75%
2. Tinnitus+Ear Blockage	59	24.18%
3. Tinnitus+Vertigo	40	16.39%
4. Tinnitus+Ear Block -age+Vertigo	28	11.48%
5. Vertigo	04	01.64%
6. Vertigo+Ear Blockage	12	04.92%
7. Ear Blockage	65	26.64%

In 2001 Gianoli observed 44% had improvement by average 15.2 dB and stratified by age, younger patients tended to have more fovorable result than older

Group A Oral steroids + methyl cobalamine 1500 microgram doses total 89					
Age Group	Cure Gr/I	Gr IIa	Gr IIB	No Cure	Total
10-20	5	3	2	2	12
20-40	7	7	6	5	25
40-71	9	14	11	18	52
Total	**21**	**24**	**19**	**25**	**89**

Table-IV

patients[14] while Kopke in 2001 observed 83% improvement by using microcatheter continuous infusion of methyl prednisolone 62.5 mg/ml delivered 14 days at a rate of 10 micro litre per hour.[15]

Xenellis observed 47% improvement in PTA of at least 10 dB by infiltrating methyl prednisolone 40 mg/ml with 21 gauge needle four times over 15 days.[16] We have used methyl prednisolone infiltration by posterior inferior quadrent grommet insertion 5 drops of methyl prednisolone 40 mg/ml. 2 time a day over 4 weeks and observed an overall (52.9%) success rate which is identical with

Group B Oral steroids + intra tympanic infiltration methyl prednisolone + methyl cobalamine total 59					
Age Group	Cure Gr/I	Gr IIa	Gr IIB	No Cure	Total
10-20	1	1	-	1	3
20-40	6	7	10	5	28
40-70	5	8	10	5	28
Total	**12**	**16**	**20**	**11**	**59**

Table-V

Xenellis and Gianoli but Kopke have observed extremely promising results.

The result in vertigo cases was poor in our series. Ben David J et had observated similar results .[17] The best result was observed with oral corticosteroid and intratympanic infiltration of methyl prednisolone + methyl cobalamine 1500 mcg. Addition of acyclovir

Group C Oral corticosteroids +intra tympanic methyl prednisolone + methyl cobalamine + acyclovir total 96					
Age Group	Cure Gr/I	Gr IIa	Gr IIB	No Cure	Total
10-20	9	9	4	1	23
20-45	8	10	18	6	42
45-70	7	11	6	7	31
Total	**24**	**30**	**28**	**14**	**96**

Table-VI

was of not much advantage except in 10-20 years age group where viral etiology may be

a factor leading to deafness.

CONCLUSION

From this study it can be concluded results are better if the patient reports, early lesser is the severity of deafness better is the prognosis. Intra tympanic infiltration of methyl prednisolone may add up prognostic rate. Acyclovir may be worth considering in selected cases where viral etiology is suspected. Intratympanic methyl prednisolone through ventilation tube improves success rate with no side affects of systemic or oral corticosteroids specifically in diabetes, hypertension and thyroid disorder patients.

REFERENCES

1. Wilson W R. Why treat sudden hearing loss? *Am J Otol* 1984; 5:481-483.

2. Okamoto M, Shitara T, Nakayama M,. Sudden deafness accompanied by asymptomatic mumps. *Acta Otolaryngol Suppl*. 1994; 514:45-8 [Medline]

3. Schuknecht HF, Donovan ED. The pathology of idiopathic sudden sensorineural haring loss. *Arch Otorhinolaryngol* 1986; 243:1-15.

4. Toubi E, Ben-David J, Kessel A, Halas K, Sabo E, Luntz M. Immune-mediated disorders associated with idiopathic sudden sensorineural hearing loss. *Ann Otol Rhinol Laryngol*. Jun 2004; 113 (6):445-9 [Medline].

5. Simmons FB. Theory of membrane breaks in sudden hearing loss. *Arch Otolaryngol*. Jul 1968; 88 (1):41-8 [Medline].

6. Gussen R. Sudden hearing loss associated with cochlear membrane rupture. Two human temporal bone reports. *Arch Otolaryngol*. Oct 1981; 107 (10):598-600. [Medline]

7. Davis H Silverman SR. *Hearng and deafness*, 4th ed. New York; Holt, Rinehart & Winston, 1978.

8. Flavia Alncar de Barros, Norma Oliveira Penido, Hugo Valter Lisboa Ramos, Maura Ligia Sanchez, Yotaka Fukuda, Audiological Evaluation of Twenty Patients Receiving Pentoxifylline and Prednisone After Sudden Deaness: Prospective Study. *International Tinnitus Journal* 2003; Vol. 9, No. 1, 17-22.

9. Hirofumi Harada Toshihiko Kato, Prognosis for Sudden Sensorineural Hearing Loss: A Retrospective Study Using Logistical Regression Analysis. *International Tinnitus Journal*, 2005; Vol. 11, No. 2, 115-118.

10. Nakashima T, Tanabe T, Yanagita N,. Risk factors for sudden deafness: A case-control study. *Auris Nasus Larynx* 1997 24(3):265-270.

11. Kronenberg J, Almagor M, Bendet E, Kushnir D. Vasoactive therapy versus placebo in the treatment of sudden hearing loss: A double blind clinical study. *Laryngoscope* 1992; 102:65-68.

12. Molini E, Serafini G, Altissimi G,. Sudden idiopathic hearing loss. Case reports in the course of ten years. *Acta Otorhinolaryngol Ital* 1998; 18(4):218-227.

13. Parnes LS, Sun AH, Freeman DJ. Corticosteroid pharmacokinetics in the inner ear fluids: an animal study followed by clinical application. *Laryngoscope*. 1999;109:1–17. [PubMed]

14. Gianoli GJ, Li JC. Transtympanic steroids for treatment of sudden hearing loss. *Otolaryngol. Head Neck Surg.* 2001;125:142–146. [PubMed]

15. Kopke RD, Hoffer ME, Wester D, O'Leary MJ, Jackson RL. Targeted topical steroid therapy in sudden sensorineural hearing loss. Otol. Neurotol. 2001;22:475–479. [PubMed]

16. Xenellis J, Papadimitriou N, Nikolopoulos T,. Intratympanic steroid treatment in idiopathic sudden sensorineural hearing loss: a control study. *Otolaryngol Head Neck Surg*. 2006;134:940–945. [PubMed]

17. Ben David J, Luntz M, Podoshin L,. Vertigo as a prognostic sign in sudden sensorineural hearing loss. *Int Tinnitus J* 2002 8:127-128.

Dr. M.K. Taneja,
Editor Indian Institute of Ear Diseases, E-982,
C.R. Park, New Delhi-110019